The first article mentioning such a link has been published in May 2004 by the *American Journal of Clinical Nutrition* (Chiu, et al. 2004). The study had enrolled 126 healthy, glucose-tolerant subjects and results have shown that 25-hydroxy-vitamin D levels were positively correlated with *insulin sensitivity* index, leading to the following conclusion: “Subjects with hypovitaminosis D are at higher risk of *insulin resistance* and the *metabolic syndrome*” (Chiu, et al. 2004). Data have quickly started to accumulate soon after. An article published in May 2005 by the *European Journal of Clinical Investigation* tells us how clinical trials and observational studies demonstrate that *calcium* and *vitamin D* deficiencies increase the risk of malignancies, of chronic inflammatory and autoimmune diseases, and of metabolic disorders such as *hypertension* and *metabolic syndrome* (Peterlik and Cross 2005). Compared to the traditional vitamin D functions in bone health and in mineral fixation, a much more widespread role has been attributed to *vitamin D* as early as in 2006, due to the identification of vitamin D receptors in plenty of other tissues (Martini and Wood 2006).

Since 2007, multiple articles have flourished about *vitamin D* deficiency, *metabolic syndrome*, *obesity*, and *diabetes* type 2. *Vitamin D* and *calcium* insufficiency may negatively influence *glycemia* (Pittas, et al. 2007). 25-hydroxy-vitamin D level is inversely associated with 10-year risk of *hyperglycemia*, with *insulin resistance*, and with *metabolic syndrome* (Forouhi, et al. 2008). Low-circulating *vitamin D* concentrations may be associated with an increased prevalence of *metabolic syndrome* (Reis, et al. 2007). This last study also suggests a link between an increased risk of *metabolic syndrome* and elevated *parathormone* levels in older men, knowing that parathormone levels generally provide the mirror image of vitamin D levels as this is how the body tries to deal with the resulting lack of *calcium* (Reis, et al. 2007).

A specific study has been carried out in 2007 to evaluate the increased prevalence of vitamin D deficiency among morbidly obese patients (body mass index of 40+). It shows that 61 % of morbidly obese patients presenting a *metabolic syndrome* suffer from *vitamin D* deficiency compared to 33 % of those who did not achieve the criteria for metabolic syndrome (Botella-Carretero, et al. 2007). Another study published in 2007 concerning 217 obese children where 25-hydroxy-vitamin D levels correlated negatively with *body mass index*; more than half of the obese children were *vitamin D* insufficient and almost half of the insufficient group suffered from severely low *vitamin D* levels (≤ 10 ng/ml) (Smotkin-Tangorra, et al. 2007). In February 2008, a huge epidemiological study collected from 6,810 British subjects aged 45 has shown an inverse association between *vitamin D* levels and *metabolic syndrome* (Hypponen, et al. 2008).

Identical findings have been published in July 2009 among 3262 middle-aged and elderly (50-70) Chinese individuals: low *vitamin D* levels are significantly associated with *metabolic syndrome* or *insulin resistance* (Lu, et al. 2009). In February 2010, another survey performed among 324 middle-aged Korean subjects led to exactly the same conclusions, i.e. a strong inverse association of serum *vitamin D* levels with both *metabolic syndrome* and *hypertension* (Kim, et al. 2010). In June 2010, a new study has provided the same conclusions after measuring the 25-OH *vitamin D* level in 542 Arab Americans where insufficiency is associated with *metabolic syndrome* and *insulin resistance* (Pinelli, et al. 2010).
Taking into account the emerging role of vitamin D in glucose homeostasis and insulin release, we cannot be surprised by the accumulation of "observational data strongly support[ing] the role of vitamin D deficiency in the pathogenesis of type 2 diabetes" (Chowdhury, et al. 2009). Therefore, as the relationship may be mediated at least partially through incident diabetes type 2, low serum concentrations of vitamin D have been associated with increased risk for cardiac events (Penckofer, et al. 2008). According a publication issued in January 2009, prospective cohort studies suggest that 25-OH-vitamin D deficiency is associated with cardiovascular diseases and with mortality over follow-up (Michos 2009).

This has been confirmed through another study published in April 2009 by the Saudi Medical Journal and conducted on 119 type 2 diabetic patients from Iran, showing a strong link between vitamin D deficiency and high body mass index ($p = 0.003$), metabolic syndrome ($p = 0.05$), and increased highly sensitive C-reactive protein ($p = 0.009$) (Bonakdaran and Varasteh 2009). Linking further on vitamin D levels and cardiovascular disease, a study has been published in July 2010 showing the positive association between vitamin D (as well as parathormone) disruption and carotid intima-media thickness (as well as metabolic syndrome prevalence) (Richart, et al. 2010). In a meta-analysis published in March 2010, ten observational studies and nine randomized control trials concerned with the association between vitamin D levels and blood pressure have been identified; eight observational studies and three randomized control trials supported an inverse association between vitamin D and blood pressure, which shows the need for more research (Feneis and Arora 2010).

As vitamin D deficiency appears to be highly prevalent and because of the purported links with diabetes as well as with cardiovascular disease, correcting vitamin D levels in order to prevent and even treat diabetes represents a "promising field to explore" (Baz-Hecht and Goldfine 2010). Already a few clinical trials suggest beneficial impact of vitamin D supplementation in prediabetes, such as improved insulin secretion and sensitivity, but most of these studies present significant limitations and it is still a little bit too early to provide evidence-based recommendations (Barengolts 2010).

Some authors even suggest that it would be possible to reverse the increasing epidemics of obesity by improving the vitamin D status as they consider that metabolic syndrome represents the expression of a "winter metabolism" leading to the accumulation of fat mass, certainly an interesting concept of human hibernation (Foss 2009). A meta-analysis published in March 2010 has shown that high levels of vitamin D among middle-age and elderly populations are strongly associated with significant decreases in cardiovascular disease, in type 2 diabetes and in metabolic syndrome (Parker, et al. 2010). If that relationship proved to be causal, treating those populations with vitamin D has a tremendous potential.

A Chinese team has recently found that supplementing 1,25-dihydroxy-vitamin D improved the insulin resistance in muscle cells, but of course these conclusions only apply in vitro, at least for the moment (Zhou, et al. 2008). Despite the limitations of our current knowledge, we can only agree with this article published in May 2010: "increasing evidence suggests that the provision of a simple, well-tolerated, and inexpensive correction of vitamin D insufficiency favorably affects the morbidity and mortality of cardiovascular disease along with the prevention of the most common chronic degenerative diseases" (Mascitelli, et al. 2010). Indeed, some data already support a possible role of vitamin D insufficiency in Parkinson disease (Evatt, et al. 2008). We therefore agree with Penckofer's title: “Let the sunshine in”!